

1 RJSP-2018-0792 Revision 1

2

3 **The effect of inspiratory muscle fatigue on acid-base status and performance**
4 **during race-paced middle-distance swimming**

5

6 Inspiratory muscle fatigue, swimming and acid-base status

7

8 Mitch Lomax¹, Jernej Kapus², Samuel Webb¹, Anton Ušaj²,

9

10 Mitch Lomax

11 Department of Sport and Exercise Science

12 University of Portsmouth

13 Cambridge Road

14 Portsmouth

15 Hampshire

16 UK

17 Telephone: +44(0)23 9284 5297

18 Email: mitch.lomax@port.ac.uk

19 ORCID id: orcid.org/0000-003-1381-0398

20 Corresponding author

21

22 Jernej Kapus

23 University of Ljubljana

24 Faculty of Sport

25 Gortanova 22

26 Ljubljana

27 Slovenia
28 Telephone: ++386 1 520 77 96
29 Email: nejc.kapus@fsp.uni-lj.si
30 ORCID id: orcid.org/0000-0002-2423-1917

31

32 Samuel Webb
33 Department of Sport and Exercise Science
34 University of Portsmouth
35 Cambridge Road
36 Portsmouth
37 Hampshire
38 UK

39 Telephone: +44(0)23 9284 5297
40 Email: samuel.webb@myport.ac.uk

41

42 Anton Ušaj
43 University of Ljubljana
44 Faculty of Sport
45 Gortanova 22
46 Ljubljana
47 Slovenia

48 Telephone: ++386 1 520 77 82
49 Email: anton.usaj@fsp.uni-lj.si

50

51 Accepted for publication 18 January 2019, Journal of Sports Sciences

52

53 RJSP-2018-0792 Revision 1

54

55

56 **The effect of inspiratory muscle fatigue on acid-base status and performance**
57 **during race-paced middle-distance swimming**

58

59 Inspiratory muscle fatigue, swimming and acid-base status

60

61 Key words: stroke rate, breathing frequency, acidosis

62

63 Manuscript word count: 3754 (excluding abstract, references, tables, figures)

64

65

66

67

68

69

70

71

72

73

74

75

76

77

78

79

80

81

82

83 **Abstract**

84

85 The aim of this study was to investigate the effect of pre-induced inspiratory muscle
86 fatigue (IMF) on race-paced swimming and acid-base status. Twenty-one collegiate
87 swimmers performed two discontinuous 400-m race-paced swims on separate days,
88 with (IMF trial) and without (control trial) pre-induced IMF. Swimming
89 characteristics, inspiratory and expiratory mouth pressures, and blood parameters were
90 recorded. IMF and expiratory muscle fatigue ($P < 0.05$) were evident after both trials
91 and swimming time was slower ($P < 0.05$) from 150-m following IMF inducement.
92 Pre-induced IMF increased pH before the swim ($P < 0.01$) and reduced bicarbonate (P
93 < 0.05) and the pressure of carbon dioxide (PCO_2) ($P < 0.05$). pH ($P < 0.05$),
94 bicarbonate ($P < 0.01$) and PCO_2 ($P < 0.05$) were lower during swimming in the IMF
95 trial. Blood lactate was similar before both trials ($P > 0.05$) but was higher ($P < 0.01$)
96 in the IMF trial after swimming. Pre-induced IMF induced respiratory alkalosis,
97 reduced bicarbonate buffering capacity and slowed swimming speed. Pre-induced and
98 propulsion-induced IMF reflected metabolic acidosis arising from dual role breathing
99 and propulsion muscle fatigue.

100

101

102

103

104

105

106

107

108 **Introduction**

109 Reducing the force generating capacity of the inspiratory muscles by 20-31% before
110 exercise decreases the duration of both exhaustive cycle ergometry (Mador &
111 Acevedo, 1991; Wüthrich et al., 2013) and fatiguing calf plantar flexion exercise
112 (McConnell & Lomax, 2006). The curtailment in performance might be the result of
113 evoking the inspiratory muscle metaboreflex, (McConnell & Lomax, 2006; Sheel et
114 al., 2001). Activation of the inspiratory muscle metaboreflex causes a
115 sympathoexcitatory increase in vascular resistance via vasoconstriction of vascular
116 beds in both inactive tissue (Sheel et al., 2001) and active muscle (Rodman,
117 Henderson, Smith & Dempsey, 2003). This could elevate blood lactate and lower pH
118 because of the negative consequences that high breathing loads have on oxygen supply
119 to, and metabolite removal from, working muscle (Harms et al., 1997; Harms, Wetter,
120 St Croix, Pegelow & Dempsey, 2000).

121

122 It is also likely that breathlessness will increase in the presence of inspiratory muscle
123 fatigue (IMF) resulting in tachypnoea at rest (Gallagher, Hof & Younes, 1985;
124 Śliwiński, Yan, Gauthier & Macklem, 1996), during cycle (85-90 % of maximal
125 power) ergometry (Mador & Acevedo, 1991; Śliwiński et al., 1996), and during
126 recovery from fatigue (Gallagher et al., 1985). The increase in breathing frequency is
127 aimed at minimizing dyspnoea (Gallagher et al., 1985; Śliwiński et al., 1996) and may
128 (Gallagher et al., 1985; Mador & Acevedo, 1991) or may not (Śliwiński et al., 1996)
129 be associated with a fall in tidal volume. The inducement of IMF can also increase the
130 phasic and tonic activity of the abdominal muscles. As this permits a more efficient
131 force-length relationship of the diaphragm, less inspiratory muscle activation is
132 required for a given degree of muscle shortening (Śliwiński et al., 1996).

133

134 Propulsion induced inspiratory muscle fatigue (IMF), which has been documented in
135 all four swimming strokes (Lomax, Iggleden, Tourell, Castle & Honey, 2012) and
136 distances ranging from 100-m to 400-m (Brown & Kilding, 2011; Lomax &
137 McConnell, 2003; Thomaidis et al., 2009), has been shown to increase breathing
138 frequency (f_r) and stroke rate (SR) during constant velocity sub-maximal swimming
139 (Lomax & Castle, 2011). Tachypnoea at a given velocity is disadvantageous, even if
140 it reduces dyspnoea, because breathing pattern is constrained by stroke mechanics and
141 increasing f_r disrupts streamlining (Seifert, Chollet & Allard, 2005). However,
142 reducing f_r to avoid such disruption is not without problem. The natural stroke-induced
143 reduction in f_r during front crawl is associated with hypercapnia and hence respiratory
144 acidosis. As well as curtailing swimming distance (Kapus, Ušaj, Kapus & Štrumbelj,
145 2003), hypercapnia is also capable of impairing respiratory muscle function (Jonville
146 et al., 2002; Rafferty et al., 1999). In addition, tidal volume increases to compensate
147 for the lower f_r (Rodriguez, 2000) thereby increasing the elastic load of breathing
148 leading to IMF (Jakovljevic & McConnell, 2009).

149

150 It is currently unknown whether IMF in swimming is partly due to respiratory acidosis
151 arising from a constrained breathing pattern, and whether f_r increases following its
152 occurrence to relieve dyspnoea or maintain normocapnia. This is complicated further
153 by the fact that some breathing muscles during swimming are also key in generating
154 forward propulsion and upper body stabilisation during the front crawl arm stroke
155 (Lomax, Tasker & Bostanci, 2014; Lomax, Tasker & Bostanci 2015; Nuber, Jobe,
156 Perry, Moynes & Antonelli, 1986; Pink, Perry, Browne, Scovazzo & Kerrigan, 1991)
157 and could therefore contribute to IMF independently of breathing. Pre-inducing IMF

158 before swimming could therefore be used to distinguish the effects of IMF on acid-
159 base status and stroke mechanics more clearly. It is conceivable that pre-existing
160 fatigue of these dual role muscles could lead to sustained metabolic perturbations
161 during subsequent swimming similar to that occurring following the inducement of
162 the inspiratory muscle metaboreflex (McConnell & Lomax, 2006) thereby curtailing
163 swimming performance.

164

165 The aim of this study was to investigate the effect of pre-induced IMF on acid-base
166 status during middle-distance race-paced swimming and its impact on stroke
167 characteristics. We hypothesised that swimming with pre-induced IMF would
168 exacerbate the metabolic perturbations and acidosis during subsequent swimming,
169 increase SR and f_r , and slow swimming time.

170

171 **Methods**

172 *Experimental approach to the problem*

173 Swimmers completed one pulmonary familiarisation and three swimming trials in an
174 indoor heated (mean \pm SD: 27.2 ± 0.3 °C) 25-m swimming pool. The first trial was
175 used to determine maximum-effort 400-m front crawl swimming time. The remaining
176 two trials were administered in a counterbalanced order and consisted of a single 400-
177 m broken (8×50 -m) front crawl swim. One of the swims was preceded by the
178 inducement of IMF (IMF trial) and the other was not (control trial). Swimming
179 characteristics (SR, f_r , swim time) were recorded during each swim and maximal
180 inspiratory and expiratory mouth pressures (P_Imax and P_Emax, respectively), pH,
181 bicarbonate (HCO_3^-), partial pressures of oxygen and carbon dioxide (PO₂ and PCO₂,

182 respectively) and blood lactate (b[lac]) were measured pre, mid (except PEmax and
183 b[lac]) and after each experimental swim from the earlobe.

184

185 *Participants*

186 Twenty-one well-trained club level swimmers volunteered for this study (see Table 1
187 for their descriptive characteristics). All provided written informed consent and
188 testing conformed to the Declaration of Helsinki. Institutional ethical approval was
189 received before the start of the study.

190

191 **Table 1 about here**

192

193 *Pulmonary familiarisation*

194 PImax (measured from residual volume) and PEmax (measured from total lung
195 capacity) were determined with the nose occluded and using a hand-held respiratory
196 pressure meter whilst standing on poolside (RPM, Micro Medical Ltd, Kent, UK).
197 Reliability was deemed present when the three highest manoeuvres from a series of
198 manoeuvres were within 10 cmH₂O. Forced vital capacity (FVC) and forced expired
199 volume in the first second of exhalation (FEV₁) were recorded for descriptive purposes
200 using a digital spirometer (Micro Spirometer, Micro Medical Ltd, Kent, UK).
201 Participants completed a minimum of three satisfactory manoeuvres with the highest
202 recorded.

203

204 *Swimming trials*

205 All swimming trials were initiated from a push start, completed using the front crawl
206 stroke, and occurred on separate days. The time taken to complete the maximum effort
207 (race-paced) 400-m swim was used to determine the pace and hence target times for
208 the two subsequent broken 400-m swimming trials, which consisted of 8 x 50-m at
209 400-m race pace. Each 50-m partial was separated by 10 s of rest, with the exception
210 that a 60 s rest separated the 4th and 5th 50-m partial so that P_Imax could be measured
211 and a capillary blood sample taken (modified from Aujouannet, Bonifazi, Hintzy,
212 Vuillerme & Rouard, 2006).

213

214 P_Imax was assessed before the inducement of IMF in the IMF trial (baseline₁), before
215 the 1st 50-m partial (baseline₂), following the 4th 50-m partial (mid-swim) and after the
216 final 50-m partial (end-swim). P_Emax was assessed at baseline₂ and end-swim only.
217 Additionally, in a sub-set of 13 participants, b[*lac*], pH, HCO₃⁻, PO₂ and PCO₂ were
218 measured from a capillary blood sample (60-80 µl) taken by micro puncture from a
219 hyperaemic earlobe and collected in heparinized glass capillaries. b[*lac*] was diluted
220 (LKM41 lactate solution, Dr. Lange, Berlin, Germany) and analysed using a
221 photometer (MINI8, Dr. Lange, Berlin, Germany) at baseline₂ and end-swim. pH,
222 HCO₃⁻, PCO₂ and PO₂ were analysed using a blood gas analyser (ABL5, Radiometer,
223 Copenhagen, Denmark) at baseline₁, baseline₂, mid-swim and end-swim.
224 Additionally, dyspnoea (Borg CR10 scale) was assessed before and at end-swim.

225

226 To measure clean swimming SR and *fr*, a 12.5-m zone was identified from the middle
227 of the pool and stroke characteristics were measured in this segment only. SR was
228 determined as the number of strokes divided by the time taken to swim through the

229 12.5-m zone (Hz, measured using a stop watch) and multiplied by 60 to convert to
230 cycles·min⁻¹ (Lomax et al., 2013). *fr* was calculated as the number of times the head
231 turned during the 12.5-m zone divided by time taken to cover the 12.5-m zone
232 multiplied by 60 (Lomax et al., 2013). The values were then averaged over the first
233 and second 25-m length of each 50-m partial to give a mean value per 50-m partial.
234 Additionally, the time taken (s) to complete each 50-m partial was recorded using a
235 stop watch.

236

237 *Inspiratory muscle fatigue inducement*

238 IMF was induced using a commercially available inspiratory muscle trainer
239 (POWERbreathe, HaB International, Southam, UK). With the nose occluded,
240 participants generated 70% of their P_Imax using a duty cycle of 0.60 (three seconds
241 for inspiration and two seconds for expiration) and an *fr* of 12. Participants coordinated
242 breathing pattern with a metronome and continued with this pattern until it could no
243 longer be maintained for three consecutive breaths. Participants then continued for a
244 further minute after which P_Imax was re-assessed and recorded as baseline₂. This has
245 been shown to result in a fall in P_Imax of 17-25% in swimmers (Lomax & Castle,
246 2011; Lomax Tasker & Bostanci, 2014), which is consistent with the magnitude (8-
247 29%) observed following 100-m-400-m front crawl swimming (Brown & Kilding,
248 2011; Jakovljevic & McConnell, 2009; Lomax & McConnell, 2003; Lomax et al.,
249 2013; Thomaidis et al., 2009).

250

251 **Data analysis**

252 Data were assessed for normality using a Shapiro-Wilk test. The impact of pre-induced
253 IMF on P_Imax, b[_{lac}], pH, HCO₃⁻, PO₂, PCO₂, between baseline₁ and baseline₂ (IMF
254 trial) were assessed using paired samples t-tests. Two-way (condition x time) repeated
255 measures ANOVA assessed for differences in swim time, SR, *fr*, b[_{lac}], pH, HCO₃⁻,
256 PO₂, PCO₂, P_Imax and P_Emax between IMF and control trials between baseline₂, mid-
257 swim and end-swim. Post hoc analyses were undertaken using repeated measures
258 ANOVA's with Bonferroni adjustments and paired samples t-tests. Wilcoxon-Signed
259 rank tests were used to assess post swim dyspnoea between trials expressed as delta
260 change (Δ) from baseline₁.

261
262 Effect sizes were calculated using Cohen's *d* for parametric data, whereby 0.2 was
263 deemed small, 0.6 moderate, 1.2 large, 2.0 very large and 4.0 extremely large
264 (Hopkins, Marshall, Batterham & Hanin, 2009). For non-parametric data, effect sizes
265 were calculated as *r*, whereby *r* is the *z* score divided by the square root of the total
266 number of observations. A value of .1 is deemed small, .3 medium and .5 and above
267 large (Field, 2013). Significance was set as $P \leq 0.05$ and statistical analyses were
268 undertaken using IBS SPSS statistics version 24 (Chicago, Illinois, USA). Unless
269 otherwise stated data are presented as mean \pm SD.

270

271 **Results**

272 Pre-inducing IMF caused P_Imax to fall by 19 ± 10 cmH₂O from 130 ± 30 cmH₂O to
273 111 ± 30 cmH₂O ($P < 0.001$, $d = 0.67$). Time ($F = 15.230$, $P < 0.001$) and condition (F
274 $= 9.911$, $P = 0.08$) were both significant for P_Imax and an interaction was observed (F

275 = 3.872, $P = 0.029$). Swimming with pre-induced IMF did not cause PI_{max} to fall
276 further. When IMF was not pre-induced, PI_{max} fell by mid-swim ($P < 0.001$, $d = 0.87$)
277 but did not fall any further by end-swim. PE_{max} was unaffected by condition but was
278 lower after swimming in both trials ($F = 24.704$, $P < 0.001$; Figure 1).

279

280 **Figure 1 about here**

281

282 Swimming time slowed during the latter part of each trial ($F = 18.827$, $P < 0.001$) and
283 there was an interaction between time and condition ($F = 3.579$, $P = 0.001$; Table 2).
284 SR and fr were affected by condition (SR: $F = 29.291$, $P < 0.001$; fr : $F = 14.93$, $P =$
285 0.001) and time (SR: $F = 3.958$, $P = 0.044$; fr : $F = 5.820$, $P = 0.001$) but no interactions
286 were observed (Table 2).

287

288 **Table 2 about here**

289

290 Mean SR and fr were correlated in both control ($r = 0.690$, $P = 0.001$) and IMF ($r =$
291 0.675 , $P = 0.001$) trials, as were mean swimming time and SR (control: $r = -0.722$, P
292 < 0.001 ; IMF: $r = -0.762$, $P < 0.001$), and mean swimming time and fr (control: $r = -$
293 0.528 , $P = 0.014$; IMF: $r = -0.534$, $P = 0.013$). The change in PI_{max} was not correlated
294 with SR, fr or swimming time ($P > 0.05$).

295

296 The inducement of IMF increased pH ($t = -4.129$, $P = 0.001$), decreased PCO_2 ($t =$
297 4.813 , $P < 0.001$) and decreased HCO_3^- ($t = 3.498$, $P = 0.004$) from baseline₁ to
298 baseline₂, but had no impact on PO_2 ($P > 0.05$). Main effects were observed for $b[laC]$
299 (time: $F = 18.374$, $P = 0.001$; condition: 191.406 , $P < 0.001$), HCO_3^- (time: $F =$

154.314, $P < 0.001$; condition: $F = 19.563$, $P = 0.001$), PCO_2 (time: 6.218, $P = 0.007$; condition: $F = 19.138$, $P = 0.001$) and pH (time: $F = 110.016$, $P < 0.001$). Interactions were observed between time and condition for b[*lac*] ($F = 12.659$, $P = 0.004$), pH ($F = 19.930$, $P < 0.001$), HCO_3^- ($F = 4.432$, $P = 0.024$) and PCO_2 ($F = 5.381$, $P = 0.012$). A moderate correlation ($r = 0.450$, $P = 0.024$) was observed in *fr* and PCO_2 for Δ between mid- and end- swim (pooled data). PO_2 was unaffected by time or condition ($P > 0.05$) (Table 3). Dyspnoea was higher ($z = -2.910$, $P = 0.003$, $r = -0.46$) following the IMF trial (IMF trial: 8.1 ± 1.3 ; control trial: 7.4 ± 2.3).

308

309 **Table 3 here**

310

311 Discussion

312 It was our aim to assess the impact of pre-induced IMF on acid-base status during
313 middle-distance race-paced swimming and stroke characteristics. Pre-inducement of
314 IMF caused respiratory alkalosis, which complicates the interpretation of the findings.
315 Nevertheless, the main observations were: 1) pre-induced IMF exacerbated the
316 metabolic perturbations occurring during subsequent swimming; 2) IMF, whether pre-
317 induced or propulsion induced, is unlikely to be attributed to respiratory acidosis; 3)
318 pre-induced IMF slowed swimming time; and 4) *fr* increased in both trials but was not
319 correlated with IMF.

320

321 The pre-inducement of IMF caused PI_{max} to fall by 14%, which is consistent with
322 that reported in response to swimming *per se* (Jakovljevic & McConnell, 2009; Lomax
323 et al., 2012; Lomax et al., 2015; Thomaidis et al., 2009). The IMF protocol
324 significantly increased pH, reduced HCO_3^- and caused a non-significant fall in PCO_2

325 (Table 3). This meant that swimmers began the swim in a state of mild hypocapnia
326 and alkalosis. It is likely that this state developed because of hyperventilation arising
327 from the increase in breathing depth associated with the IMF protocol and not IMF
328 *per se* (Chin et al., 2007; Costanzo, 2010; Le Blanc, Parolin, Jones & Heigenhauser,
329 2002). It could therefore be argued that as the method we adopted to induce IMF
330 created a breathing pattern (hyperventilation) antithetical to that in front crawl
331 swimming (hypoventilation), it may not reflect the metabolic milieu accompanying
332 swimming-induced IMF. Indeed, the observation that swimmers began the swim in
333 the IMF trial with lower HCO_3^- levels means that their ability to buffer H^+ during
334 subsequent swimming was likely compromised. This could be exacerbated by a
335 slowing of pulmonary oxygen uptake kinetics and microvascular blood flow, which
336 have been observed in the presence of hyperventilation-induced hypocapnic alkalosis
337 (Chin et al., 2007; Chen, Heigenhauser, Paterson & Kowalchuk, 2013). Contrastingly,
338 when CO_2 is added to an inspire during hyperventilation to prevent hypocapnic
339 alkalosis, pH and PCO_2 are prevented from falling and pulmonary oxygen uptake
340 kinetics and microvascular blood flow are improved (Chin et al., 2013). Had a
341 hypercapnic gas mixture been administered during IMF inducement in the current
342 study, the resultant respiratory alkalosis would have been prevented. This approach
343 should be adopted in future.

344

345 The control trial did not suffer from this same limitation as swimmers began the
346 control trial swim with normal pH, HCO_3^- and PCO_2 values (Table 3). By mid-swim
347 (i.e. 200-m) however, P_{Imax} had fallen by 24% indicating the presence of IMF (Figure
348 1). At this same time point, pH had become mildly acidic (i.e. < 7.35), HCO_3^- levels
349 had fallen (Table 3), but there was no evidence of hypercapnia or respiratory acidosis.

350 This pattern of change in pH, HCO_3^- and PCO_2 during swimming in the control trial
351 was similar to the pattern observed in response to swimming in the IMF trial, but the
352 absolute values differed (Table 3).

353

354 Pre-inducing IMF did exacerbate the metabolic perturbations (e.g. pH, HCO_3^- , PCO_2
355 and $\text{b}[\text{lac}]$) occurring during the swim. Despite reducing the buffering capacity of H^+
356 in this trial it did not translate into a greater magnitude of IMF (Figure 1). This does
357 not mean that a lowered buffering capacity was without consequence. As the
358 progressive fall in pH and HCO_3^- (Table 4) was greater in the IMF trial, the slower
359 swimming times (Table 2) in this trial might be indicative of a reduced ability to buffer
360 H^+ generated during swimming.

361

362 Acidosis can be respiratory or metabolic in origin, or can be a combination of both
363 (Brooks et al., 2000). The progressive fall in pH without concomitant hypercapnia
364 makes it likely we detected its metabolic origin. Moreover, the development of IMF,
365 whether pre-induced or propulsion-induced, cannot be attributed to ventilatory
366 impairment causing respiratory acidosis; at least not when the breathing pattern is
367 optional and \dot{V}_E is $\geq 25 \text{ breaths} \cdot \text{min}^{-1}$. However, there are a number of caveats to this
368 interpretation.

369

370 It is known that exercise can increase carbon dioxide storage in the body and this
371 storage capacity is diminished at higher exercise intensities leading to an increase in
372 carbon dioxide excretion (Jones & Jurkowski, 1979). The relatively short duration of
373 the 50-m repeat swims and the fact that they were not maximal effort swims but were
374 instead based on 400-m race pace, could be incompatible with the detection of

375 hypercapnia. Additionally, it took approximately 20-30 s to obtain blood samples for
376 analysis during which time breathing was very intense. If a small swimming-induced
377 increase in PCO_2 did occur, it could be reversed during this period resulting in the
378 measured PCO_2 failing to change. Lastly, failure to observe hypercapnia does not
379 necessarily mean that ventilation poses no limitation during front crawl swimming, or
380 that hypercapnia does not occur. It may be that a ventilatory limitation was not
381 sufficient to cause respiratory acidosis. In support of this, Ušaj (1999) found
382 ventilation to be less effective at compensating for metabolic acidosis during 400-m
383 race-paced front crawl swimming (pH of 7.09 ± 0.09 , PCO_2 of 4.0 ± 0.5 kPa) compared
384 to maximal effort kayak paddling (pH of 7.17 ± 0.05 , PCO_2 of 3.6 ± 0.2 kPa) but this
385 failed to result in hypercapnia in the former. We observed a similar pattern and also
386 found the Δ (increase) in f_r between mid- and end- swim (mid-swim being the first
387 time point where IMF was evident in both trials) was correlated with Δ (fall) in PCO_2
388 ($r = 0.450$), suggesting that the increase in f_r , which occurred in both trials, partly
389 contributed to the prevention of hypercapnia. However, the small coefficient of
390 determination (20%) indicates that prevention of hypercapnia was not the primary
391 cause for the increase in f_r .

392

393 It has been proposed that stimulation of thin fibre afferents within fatigued inspiratory
394 muscles create an additional breathing stimulus. This increase in central respiratory
395 drive to the weakened inspiratory muscles is discernible as dyspnoea (Gallagher et al.,
396 1985; Mador & Acevedo, 1991; Sliwinski et al., 1996) and the resultant tachypnoea is
397 thought to be directed at minimising this sensation (Gallagher et al., 1985). Post swim
398 dyspnoea was higher than baseline₁ in both trials and was higher still in the IMF trial,
399 but no interaction was observed between condition and time for f_r . This lack of

400 interaction is not surprising given that no interaction was observed between time and
401 condition for f_r .

402

403 In previous studies we have shown that 20 s front crawl sprinting following the
404 inducement of IMF leads to an increase in SR but not f_r (Lomax et al., 2014) and that
405 IMF magnitude is not correlated with f_r during the four swimming strokes (Lomax et
406 al., 2012). Although f_r and SR were correlated in the current study ($P < 0.001$), the
407 coefficient of determination was only 46-48%. Thus, the cause of the increase in f_r that
408 may or may not accompany IMF in swimming is multifactorial and is likely impacted
409 by the ventilatory drive to maintain normocapnia, a need to alleviate dyspnoea, and an
410 increase in SR if SR does increase: SR did not increase in the present study but has
411 been shown to previously (Lomax & Castle, 2011).

412

413 It is important to recognise here that the breathing muscles in swimming are also key
414 propulsion muscles. For example, the pectoralis major, latissimus dorsi, upper
415 trapezius, and serratus anterior, are activated during deep inspirations (Kendall et al.,
416 2005) and front crawl swimming (Nuber et al., 1986; Pink et al., 1991). We have
417 shown that 20 s arms only front crawl sprinting is sufficient to induce pectoralis major
418 fatigue but not latissimus dorsi fatigue (Lomax et al., 2014). In contrast, latissimus
419 dorsi and pectoralis major fatigue have been reported following 100-m (63 ± 2 s) full
420 stroke front crawl swimming (Stirn et al., 2011). Given that the measurement of PImax
421 is holistic in nature, the pressure recorded at the mouth will reflect the collective
422 activity of all the muscles recruited (Gibson, 1995). This will include contributions
423 from muscles vital in creating forward propulsion and trunk stability (Pink et al., 1991)
424 and fatigue of these muscles is evident in the PImax manoeuvre (Lomax et al., 2015).

425

426 It is therefore possible that the propulsion and stabilisation requirements of the dual
427 role muscles during swimming caused fatigue of these muscles and this in-turn was
428 evident in the holistic assessment of P_Imax and P_Emax. The presence of IMF and
429 expiratory muscle fatigue therefore does not automatically indicate a breathing
430 induced origin for fatigue. Rather, it could be propulsion induced. If this is the case,
431 the aetiology of IMF in the present study differed between the two trials: breathing
432 induced in the IMF trial and propulsion induced in the control trial. However, the net
433 effect was the same: the dual role breathing and propulsion/trunk stabilisation muscles
434 experienced fatigue. As we did not measure electromyography of the dual role muscles
435 or their force output while swimming, we are unable to identify exactly which muscles
436 were affected.

437

438 **Conclusion**

439 Swimming with pre-induced IMF caused transient respiratory alkalosis, which was
440 reversed by mid-swim. Pre-induced IMF was associated with a greater level of
441 metabolic perturbation including reduced H⁺ buffering capacity, increased acidosis
442 during subsequent swimming and slowed swimming times. However, the overall
443 magnitude of IMF experienced was similar between IMF and control trials and the
444 increase in f_r accompanying both trials was not correlated with IMF but was correlated
445 with PCO₂.

446

447 The cause of acidosis and IMF, whether pre-induced or propulsion induced, could not
448 be attributed to ventilatory impairment and therefore must have been metabolic in

449 origin. Given the dual role function of the affected muscles we think it likely that IMF
450 occurring in response to swimming reflects propulsion induced fatigue during 400-m
451 crawl swimming.

452

453

454

455

456

457

458

459

460

461

462

463

464

465

466

467

468

469

470

471

472

473

474 **References**

475

476 Aujouannet, Y. A., Bonifazi, M., Hintzy, F., Vuillerme, N., & Rouard, A. H. (2006).
477 Effects of a high-intensity swim test on kinematic parameters in high-level athletes.
478 *Applied Physiology Nutrition and Metabolism*, 31, 150-158.

479

480 Brooks, G. A., Fahey, D. F., White, T. P., & Baldwin, K. M. (2000). *Exercise*
481 *Physiology*. California: Mayfield Publishing Company.

482

483 Brown, S., & Kilding, A. E. (2011). Exercise-induced inspiratory muscle fatigue
484 during swimming: the effect of race distance. *Journal of Strength and Conditioning*
485 *Research*, 25, 1204–1209.

486

487 Chin, L. M. K., Heigenhauser, G. J. F., Paterson, D. H. & Kowalchuk, J. M. (2013).
488 Effect of voluntary hyperventilation with supplemental CO₂ on pulmonary O₂ uptake
489 and leg blood flow kinetics during moderate-intensity exercise. *Experimental*
490 *Physiology*, 98.12, 1668-1682.

491

492 Chin, L. M. K., Leigh, R. J., Heigenhauser, G. J. F., Rossiter, H. B., Paterson, D. H.,
493 & Kowalchuk, J. M. (2007). Hyperventilation-induced hypocapnic alkalosis slows the
494 adaption of pulmonary O₂ uptake during the transition to moderate-intensity exercise.
495 *Journal of Physiology* 583.1, 351-364.

496

497 Field, A. (2013). *Discovering statistics using IBM SPSS statistics*. London: Sage.

498

499 Gallagher, C. G., Hof, V. I. M., & Younes, M. (1985). Effect of inspiratory muscle
 500 fatigue on breathing pattern. *Journal of Applied Physiology*, 59, 1152-1158.
 501
 502 Gibson, G. J. (1995). Measurement of respiratory muscle strength. *Respiratory*
 503 *Medicine*, 89, 529–535.
 504
 505 Harms, C. A., Babcock, M. A., McClaren, S. R., Pegelow, D. F., Nickele, G. A.,
 506 Nelson, W. B., & Dempsey, J. A. (1997). Respiratory muscle work compromises leg
 507 blood flow during maximal exercise. *Journal of Applied Physiology*, 82, 1573-1583.
 508
 509 Harms C. A., Wetter, T. J., St Croix, C. M., Pegelow, D. F., & Dempsey, J. A. (2000).
 510 Effects of respiratory muscle work on exercise performance. *Journal of Applied*
 511 *Physiology*, 89, 131-138.
 512
 513 Hopkins, W.G., Marshall, S.W., Batterham, A.M., & Hanin, J. (2009). Progressive
 514 statistics for studies in sports medicine and exercise science. *Medicine and Science in*
 515 *Sports and Exercise*, 41, 3-12.
 516
 517 Jones, N. J., & Jurkowski, J. E. (1979). Body carbon dioxide storage capacity in
 518 exercise. *Journal of Applied Physiology*, 46, 811-815.
 519
 520 Jonville, S., Delpech, N., & Denjen, A. (2002). Contribution of respiratory acidosis to
 521 diaphragm fatigue at exercise. *European Respiratory Journal*, 19, 1079-1086.
 522

523 Kapus, J., Ušaj, A., Kapus, V., & Štrumbelj, B. (2003). Influence of reduced breathing
 524 during intense front crawl swimming on some respiratory and metabolic values in
 525 blood. *Kinesiology Slovenica*, 9, 12-17.

526

527 Kendall, F. P., McCreary, E. K., Provance, P. G., Rodgers, M. M., & Romani, W. A.
 528 (2005). *Muscles, Testing and Function With Posture and Pain*. Baltimore: Williams
 529 & Wilkins.

530

531 LeBlanc, P. J., Parolin, M. L., Jones, N. L., & Heigenhauser, G. J. F. (2002). Effects
 532 of respiratory alkalosis on human skeletal muscle metabolism at the onset of
 533 submaximal exercise. *Journal of Physiology*, 544.1, 303-313.

534

535 Lomax, M., & Castle, S. (2011). Inspiratory muscle fatigue significantly affects
 536 breathing frequency, stroke rate, and stroke length during 200-m front crawl
 537 swimming. *Journal of Strength and Conditioning Research*, 25, 2691-2695.

538

539 Lomax, M., Iggleden, C., Tourell, A., Castle, S., & Honey, J. (2012). Inspiratory
 540 muscle fatigue after race-paced swimming is not restricted to the front crawl stroke
 541 *Journal of Strength and Conditioning Research*, 26, 2729-2733.

542

543 Lomax, M. E., & McConnell, A. K. (2003). Inspiratory muscle fatigue in swimmers
 544 after a single 200 m swim. *Journal of Sports Sciences*, 21, 659–664.

545

546 Lomax, M., Tasker, L., & Bostanci, O. (2014). Inspiratory muscle fatigue affects
547 latissimus dorsi but not pectoralis major activity during arms only front crawl
548 sprinting. *Journal of Strength and Conditioning Research*, 28, 2262-2269.

549

550 Lomax, M., Tasker, L., & Bostanci, O. (2015). An electromyographic evaluation of
551 dual role breathing and upper body muscles in response to front crawl swimming.
552 *Scandinavian Journal of Medicine and Science in Sports*, 25, 1-7.

553

554 Lomax, M., Thomaids, S. P., Iggleden, C., Toubekis, A. G., Tiligadas, G.,
555 Tokmakidis, S. P., Oliveira, R. C., & Costa, A. M. (2013). The impact of swimming
556 speed on respiratory muscle fatigue during front crawl swimming: a role for critical
557 velocity? *International Journal of Swimming Kinetics*, 2, 3-12.

558

559 Mador, M. J., & Acevedo, F. A. (1991). Effect of respiratory muscle fatigue on
560 subsequent exercise performance. *Journal of Applied Physiology*, 70, 2059-2065.

561

562 McConnell, A. K., & Lomax, M. (2006). The influence of inspiratory muscle work
563 history and specific inspiratory muscle training upon human limb muscle fatigue.
564 *Journal of Physiology* 577.1, 445-457.

565

566 Nuber, G. W., Jobe, F. W., Perry, J., Moynes, D. R., & Antonelli, D. (1986). Fine wire
567 electromyography analysis of muscles of the shoulder during swimming. *The*
568 *American Journal of Sports Medicine*, 14, 7-11.

569

570 Pink, M., Perry, J., Browne, A., Scovazzo, M. L., & Kerrigan, J. (1991). The normal
 571 shoulder during freestyle swimming: an electromyographic and cinematographic
 572 analysis of twelve muscles. *American Journal of Sports Medicine*, 19, 569–576.
 573

574 Ušaj, A. (1999). The characteristics of acidosis after maximal front crawl swimming
 575 on 400 m. In V. Strojnik & A. Ušaj (Ed.), *Sports Kinetics*. (pp. 398-401). University
 576 of Ljubljana, Faculty of Sport.
 577

578 Seifert, L., Chollet, D., & Allard, P. (2005). Arm coordination symmetry and breathing
 579 effort in front crawl. *Human Movement Science*, 26, 234-256.
 580

581 Rafferty, G. F., Harris, M. L., Polkey, M. I., Greenough, A. & Moxham, J. (1999).
 582 Effect of hypercapnia on maximal voluntary ventilation and diaphragm fatigue in
 583 normal humans. *American Journal of Respiratory Critical Care Medicine*, 160, 1567-
 584 1571.
 585

586 Rodman, J. R., Henderson, K. S., Smith, C. A. & Dempsey, J. A. (2003).
 587 Cardiovascular effects of the respiratory muscle metaboreflexes in dogs: rest and
 588 exercise. *Journal of Applied Physiology*, 95, 1159-1169.
 589

590 Rodriguez, F.A. (2000). Maximal oxygen and cardiorespiratory response to maximal
 591 400-m free swimming, running and cycling tests in competitive swimmers. *Journal of*
 592 *Sports Medicine and Physical Fitness*, 40, 87-95.
 593

594 Sheel, A. W., Derchak, P. A., Morgan, B. J., Pegelow, D. F., Jacques, A. J. &
595 Dempsey, J. A. (2001). Fatiguing inspiratory muscle work causes a reflex reduction
596 in resting leg blood flow in humans. *Journal of Physiology*, 537.1, 277-289.

597

598 Sliwinski, P., Yan, S., Gauthier, A. P., & Macklem, P. T. (1996). Influence of global
599 inspiratory muscle fatigue on breathing during exercise. *Journal of Applied*
600 *Physiology*, 80, 1270-1278.

601

602 Thomaidis, S. P., Toubekis, A. G., Mpousmoukilia, S. S., Douda, H. T., Antoniou, P.
603 D., & Tokmakidis, S. P. (2009). Alterations in maximal inspiratory mouth pressure
604 during a 400-m maximum effort front-crawl swimming trial. *Journal of Sports*
605 *Medicine and Physical Fitness*, 49, 194-200.

606

607

608

609

610

611

612

613

614

615

616

617

618

619

620

621

622

623 Figure 1. PImax (A) and PEmax (B) immediately before the swim (baseline₂), mid-
624 swim and end-swim in control (filled bars) and IMF (open bars) trials.

625

626 Notes: †(P < 0.01) different to control trial at given time point; *(P < 0.01) different
627 to baseline₂ within trial. See text for abbreviations.

628

629

630

631

632

633

634

635

636

637

638

639

640

641

642

643

644

645

646

647

648

649

650

651

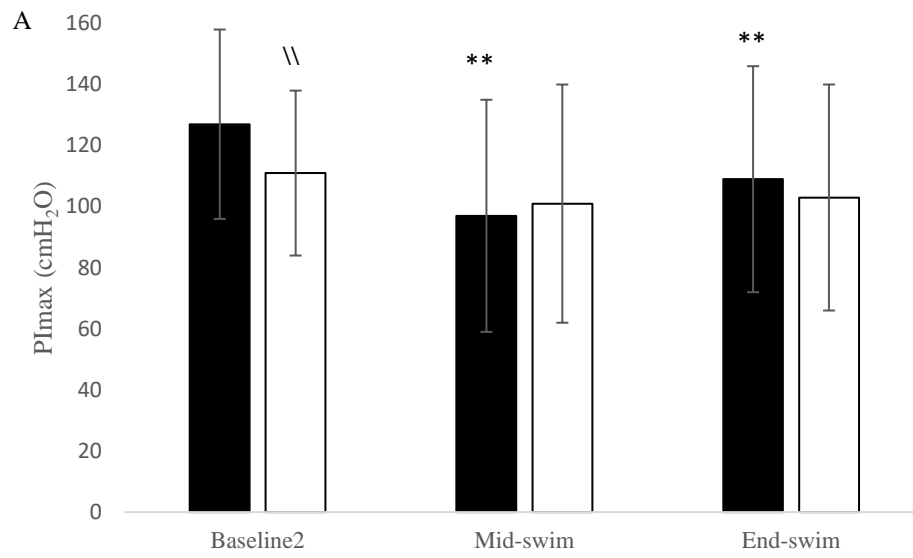
652

653

654

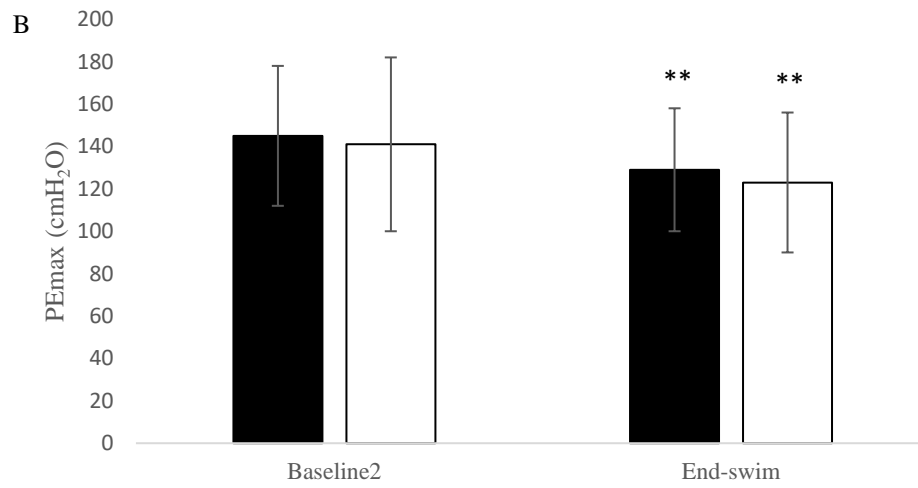
655

656



657

658



659

660

661

662

663

664

665

666

667

668

669

670

671 Table 1. Descriptive characteristic of swimmers at the start of the study: mean \pm SD

672 Measure	Group	Males	Females
673 n	21	12	9
674 Age (years)	22 \pm 3	22 \pm 3	22 \pm 3
675 Body mass (kg)	71.2 \pm 12.7	77.6 \pm 13.1	62.3 \pm 4.9**
676 Stature (m)	1.75 \pm 0.10	1.81 \pm 0.09	1.65 \pm 0.07**
677 FVC (l)	5.26 \pm 1.11	6.04 \pm 0.74	3.69 \pm 0.39**
678 FEV ₁ (l·s ⁻¹)	4.42 \pm 0.91	4.98 \pm 0.77	4.21 \pm 0.42**
679 FEV ₁ /FVC (%)	85 \pm 7	82 \pm 6	88 \pm 7
680 PImax (cmH ₂ O)	134 \pm 29	136 \pm 32	132 \pm 27
681 PEmax (cmH ₂ O)	154 \pm 36	164 \pm 42	138 \pm 24

682 Notes. **($P \leq 0.01$) different to males. See text for abbreviations.

683
684
685
686
687
688
689
690
691
692
693
694
695
696
697
698

Table 2. Swimming time, stroke rate (SR) and breathing frequency (f_r) per 50-m distance and partial number per trial: group mean \pm SD (n = 21)

50-m distance and partial number									
		50-m	100-m	150-m	200-m	250-m	300-m	350-m	400-m
Parameter	Trial	1	2	3	4	5	6	7	8
Time (s)	control	37.1 \pm 8.1	39.0 \pm 7.3**	39.7 \pm 6.7**]]	40.3 \pm 7.3**]]	38.3 \pm 7.2	39.8 \pm 7.1*]]	41.0 \pm 7.2**†]]§§	40.9 \pm 7.3**]]§§
	IMF	36.4 \pm 6.7	39.0 \pm 6.7**	40.1 \pm 6.5**]]]	40.7 \pm 7.3**]]	38.7 \pm 7.0	40.7 \pm 7.3*]]]	41.6 \pm 7.3**†]]§§]	41.5 \pm 7.6**]]§§]
SR	control	37 \pm 9	35 \pm 7	34 \pm 6]]	34 \pm 6]	36 \pm 6	35 \pm 5	35 \pm 5	35 \pm 5
(cycles·min ⁻¹)	IMF	39 \pm 8†	37 \pm 7	35 \pm 5]]	35 \pm 5]]	38 \pm 6	36 \pm 5	36 \pm 6	37 \pm 6
f_r	control	25 \pm 9	26 \pm 7§]	28 \pm 9*	27 \pm 7§§	28 \pm 7*	29 \pm 6**	28 \pm 5*	29 \pm 6*
(breaths·min ⁻¹)	IMF	26 \pm 9	29 \pm 8**	30 \pm 8**	30 \pm 7**	30 \pm 7*	31 \pm 7**	31 \pm 6**	32 \pm 7**

Note. *(P < 0.05) ***(P < 0.01) different to 1st 50-m; †(P < 0.05) different to 2nd 50-m;](P < 0.05)]](P < 0.01) different to 5th 50-m; §(P < 0.05) §§(P < 0.01) different to 6th 50-m;]](P < 0.05)]](P < 0.01) different to control at time point. See text for abbreviations.

Table 3. Blood parameters measured before, mid-swim (200-m) and end-swim (400-m) per trial: group mean \pm SD (n = 13)

Parameter	Trial	Baseline ₁	Baseline ₂	Mid-swim	End-swim
b[lac] (mmol·l ⁻¹)	control	/	1.4 \pm 0.2	/	8.2 \pm 2.4**
	IMF	/	1.6 \pm 0.5	/	10.1 \pm 1.9**
pH	control	/	7.44 \pm 0.03 ^{††}	7.32 \pm 0.04**	7.27 \pm 0.04** ^{††}
	IMF	7.43 \pm 0.01	7.48 \pm 0.04 ^{§§} ^{††}	7.30 \pm 0.04 ^{§§**}	7.23 \pm 0.05 ^{***§§} ^{††}
HCO ₃ ⁻ (mmol·l ⁻¹)	control	/	24.1 \pm 1.4 ^{††}	18.9 \pm 2.7**	16.4 \pm 2.7** ^{††}
	IMF	24.2 \pm 1.2	22.8 \pm 1.5 ^{§§††}	16.8 \pm 2.6 ^{***§§}	13.8 \pm 2.3 ^{***§§††}
PCO ₂ (kPa)	control	/	4.8 \pm 0.4	5.0 \pm 0.5	4.9 \pm 0.5
	IMF	4.9 \pm 0.2	4.1 \pm 0.7 ^{§§††}	4.7 \pm 0.5*	4.5 \pm 0.5
PO ₂ (kPa)	control	/	10.9 \pm 1.2	11.8 \pm 2.4	11.6 \pm 1.9
	IMF	11.9 \pm 2.5	11.5 \pm 3.7	12.0 \pm 1.9	11.7 \pm 1.2

Note. [§](P < 0.05) ^{§§}(P < 0.01) different to baseline₁ (within-trial comparison with baseline₂); *(P < 0.05) **(P < 0.01) different to baseline₂; [†](P < 0.05) ^{††}(P < 0.01) different to mid-swim; _{||}(P < 0.05) _{|||}(P < 0.01) different to control trial at given time point. See text for abbreviations.